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Original Article

Exercise capacity in the Fontan circulation

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Abstract The Fontan operation can create a stable circulation from childhood through early adulthood. However, the absence of a sub-pulmonary pumping chamber leads to a physiology in which exercise capacity is limited and decreases with age starting in adolescence. The limitation in exercise capacity is more pronounced at peak levels of exercise, but is still present during more modest levels of activity. The underlying causes of exercise impairment relate to both central cardiovascular factors (oxygen delivery) and peripheral factors (oxygen extraction). Interventions to improve cardiac preload and to improve lean muscle mass may help to improve exercise capacity and, perhaps, will alter the "natural history" of the progressive decline.

Keywords: Congenital heart disease; exercise; Fontan

The Fontan operation has been the standard of care for the surgical palliation of children with univentricular congenital heart disease for nearly four decades.[1,2] Although this procedure can create a stable physiology through early adulthood, there are inherent limitations related to the loss of a sub-pulmonary pumping chamber and the absence of pulsatile flow in the pulmonary arteries. The direct connection of the inferior and superior venae cavae to the pulmonary arteries results in a circulation characterised by chronic low cardiac output and elevated central venous pressure. These abnormalities are generally well tolerated for a number of years, but result in impairment in exercise capacity starting from a very young age.

Poor exercise performance, objectively measured by decreased maximal minute oxygen consumption (VO_2), is well documented in Fontan patients.[3–8] Studies have consistently demonstrated that average maximal VO_2 in Fontan patients is 60–65% of predicted value for age and gender.[8,9] In a landmark cross-sectional analysis of 411 Fontan patients, the Pediatric Heart Network Investigators demonstrated a population mean maximal VO_2 of 26.3 ± 6.9 ml/kg/min, about 65% predicated value for age and gender. However, there was considerable population variability – maximal VO_2 ranged from 19 to 112% predicted value for age and gender. Some patients performed well, even above the norm, whereas others had severely diminished exercise capacity.[7] Overall, only 28% of the participants performed within the normal range for age and gender. Although longitudinal data are not yet available on this cohort, smaller longitudinal studies have demonstrated a progressive decline in the exercise performance of young adults, as measured by maximal VO_2, of ~2.6%/year.[9]

Other measures of exercise capacity, such as maximal physical working capacity or maximal power, are depressed to a similar degree as maximal VO_2. Interestingly, certain other measures of exercise performance are less impaired. VO_2 at the anaerobic threshold is significantly better as a percentage of predicted at ~75%. The reason for this will be discussed below. In addition to alterations in metabolic measurements, impaired chronotropic performance is also observed as a universal finding in all large studies of exercise in Fontan patients. Maximal heart rates tend to cluster

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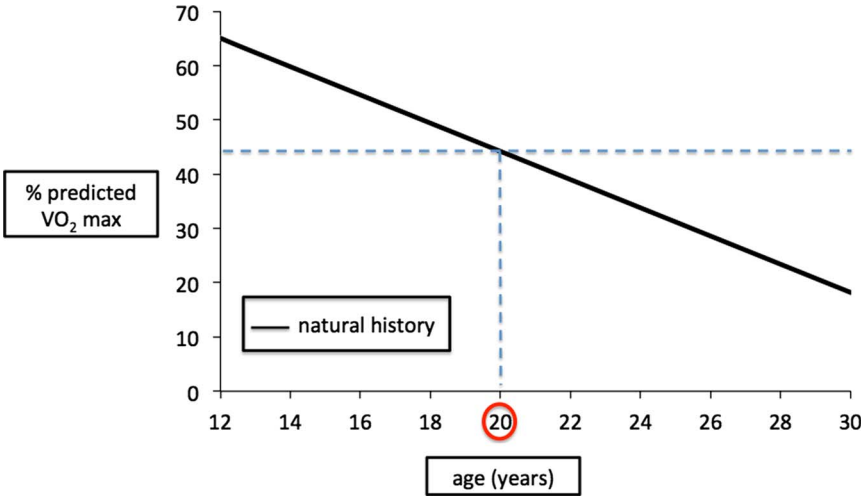


Figure 1.
At the onset of puberty, exercise performance is typically in the range of 65% predicted for age and gender. This gradually declines at a rate of ~2.6%/year such that exercise capacity would be expected to fall below the threshold for symptomatic heart failure sometime towards the end of the second decade.

around 155 to 165 bpm in almost all studies. This chronotropic response is most likely a consequence of the factors that limit exercise performance in Fontan physiology rather than a primary cause of exercise impairment.

The slow, steady decline in exercise capacity experienced by patients with Fontan physiology has important implications for their long-term outcome. Once maximal exercise capacity drops below a threshold of about 50% predicted for age and gender, symptomatic heart failure is more prevalent.[10,11] Given the baseline exercise capacity identified in the PHN study, and the rate of decline identified by Giardini et al in their longitudinal study, patients with Fontan physiology can expect to fall below the crucial threshold sometime in the early portion of their third decade of life (Fig 1). In a recent study of 321 young adults with Fontan physiology at an average age of 21, the baseline mean maximal VO_2 was just 52% predicted. As one might expect, in a 21-month follow-up period 41% of these patients required hospitalisation for heart failure, whereas 9% either died or underwent heart transplantation.[10] These findings are sobering for anyone involved in the care of patients with Fontan physiology. They point to the urgent need to develop a more thorough understanding of the limitations of the Fontan circulation and to develop potential interventions to address the deficiencies.

Exercise performance: central and peripheral factors

Exercise impairment in children with Fontan physiology may be due to both central cardiovascular factors (oxygen delivery) and peripheral skeletal muscle factors (oxygen extraction). Although central factors have been recognised to impact Fontan exercise capacity, recent attention has focused on the importance of peripheral "non-cardiac" factors. Lean muscle mass and focused training are important modulators of exercise capacity in both congenital and adult-onset heart disease, and may be important to Fontan exercise capacity as well.

Central cardiovascular factors

The PHN cross-sectional study assessed the relative contributions of central cardiovascular factors including chronotropic impairment, arterial oxygen desaturation and O_2 pulse – a surrogate for stroke volume – to the variance in aerobic capacity as measured by maximal VO_2, as well as anaerobic threshold and maximal physical working capacity. Although chronotropic impairment and arterial desaturation were common, they contributed little to variance in exercise capacity seen in this cohort (1 and 3%, respectively). The primary central cardiovascular determinant of low VO_2 was impaired stroke volume, accounting for 73% of the per cent-predicted maximal VO_2, as well as 25 to 35% of the variance in physical working capacity and anaerobic threshold.[7] Without a sub-pulmonary "pump", filling of the systemic ventricle depends on passive flow of blood through the lung vasculature. This limitation of the Fontan circulation is critical during periods of increased demand, such as intense exercise.

In the absence of a sub-pulmonary ventricle, passive pulmonary blood flow depends on the pressure difference between the central venous system and the

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ventricular end-diastolic pressure, as well as relatively low resistance in the pulmonary vasculature. Given the limited ability to increase the pre-pulmonary driving pressure, any alteration in pulmonary vascular resistance or end-diastolic pressure can have a profound impact on limiting ventricular preload. Even with a relatively normal pulmonary vascular resistance and end-diastolic pressure, the inability to increase the driving force of blood flow through the pulmonary vasculature results in decreased preload and, therefore, decreased stroke volume. The limitations of Fontan physiology, although present at rest, are more pronounced during exercise. In the normal two-ventricle circulation, exercise results in an increase in right ventricular systolic pressure of up to 50 mmHg with a corresponding decrease in pulmonary vascular resistance through the nitric oxide pathway in response to increased pulsatile flow.[12,13] This allows for an increase in cardiac output of up to 500% of baseline levels. In the Fontan circulation, it is not possible to increase the pressure gradient across the pulmonary vascular bed to the degree of a two-ventricle circulation, and the absence of pulsatile flow likely eliminates the normal nitric oxide mediated response of pulmonary vascular resistance (Fig 2). In this circulation, the increase in cardiac output with exercise is often limited to ~200% of baseline.

This effect on ventricular preload is more pronounced at higher heart rates. Diastolic filling times are limited, and the inability to maintain adequate flow through the pulmonary bed results in decreased ventricular filling. The result of this is an inability to maintain stroke volume and either a flat or falling cardiac output at higher heart rates. As consequence of this physiology, aerobic capacity at higher workloads is disproportionally impaired. In the PHN study, VO_2 at the anaerobic threshold was significantly better than at maximal levels, 78 versus 65%. The ability to maintain a higher submaximal VO_2 with the Fontan physiology does have some positive benefits. Sustained physical actives that are usually performed below the anaerobic threshold will be better tolerated. These include almost all the activities of daily living. As such, patients with Fontan physiology may often be more functional than would be expected on the basis of the maximal values obtained during exercise testing.

The efficiency of the Fontan circuit itself also affects exercise performance. Subtle differences in Fontan circuit geometry – collision of flow from the cavae into the pulmonary arteries, differential pulmonary artery flow – may cause significant power loss within the Fontan pathway.[14] As flow through the Fontan increases with exercise, power loss may increase to an even greater degree in some circuit geometries. Circuits with greater power loss may be less efficient in increasing flow through the Fontan into the pulmonary arteries during times of increased demand, further limiting the ability to adequately preload the systemic ventricle during exercise. The overall contribution of this type of power loss to exercise performance is difficult to quantify. The power loss is not linear and is difficult to measure at higher cardiac outputs in vivo. However, in certain cases it is almost certainly a significant contributing factor to limiting pulmonary blood flow. Unfortunately at this time, it is essentially impossible to do pre-operative geometric modelling of the Fontan circuits that will reliably predict the power loss in a patient when they reach the age at which formal exercise testing can be undertaken. The research of this type of modelling is nevertheless promising for future surgical planning.

Peripheral factors

Although less recognised than central cardiovascular factors, peripheral factors can also play a substantial role in the limitation in exercise performance in the Fontan population. From the PHN cross-sectional study, it is clear that stroke volume is responsible for the major portion of the central variance seen in the exercise performance. However, it is also clear that central factors are responsible for only about half of the overall variance in physical working capacity and anaerobic threshold seen in this population. This is not surprising and is similar to healthy populations, as well as those with other types of both congenital and acquired heart disease.

Peripheral factors, especially muscle mass and conditioning, appear to be at least as important in determining exercise performance in this population as central factors. We are learning that children with Fontan physiology have deficits in lean mass and skeletal muscle that may impact their oxygen extraction. As a result, even when oxygen delivery is adequate, the ability of the peripheral musculoskeletal system to meet metabolic demand may be compromised.

Fontan patients are haemodynamically similar to adults with heart failure with preserved ejection fraction, the most common form of heart failure in older patients, in that abnormal ventricular preload is the predominate driving force of diminished cardiac output. Exercise intolerance is the primary chronic symptom in this population and, similar to the Fontan circulation, diminished exercise capacity may be due to both central cardiac and peripheral – skeletal muscle – factors. The contribution of lean mass deficits to decreased maximal VO_2 is well recognised in the heart failure with preserved ejection fraction population,[15] and overall deficits

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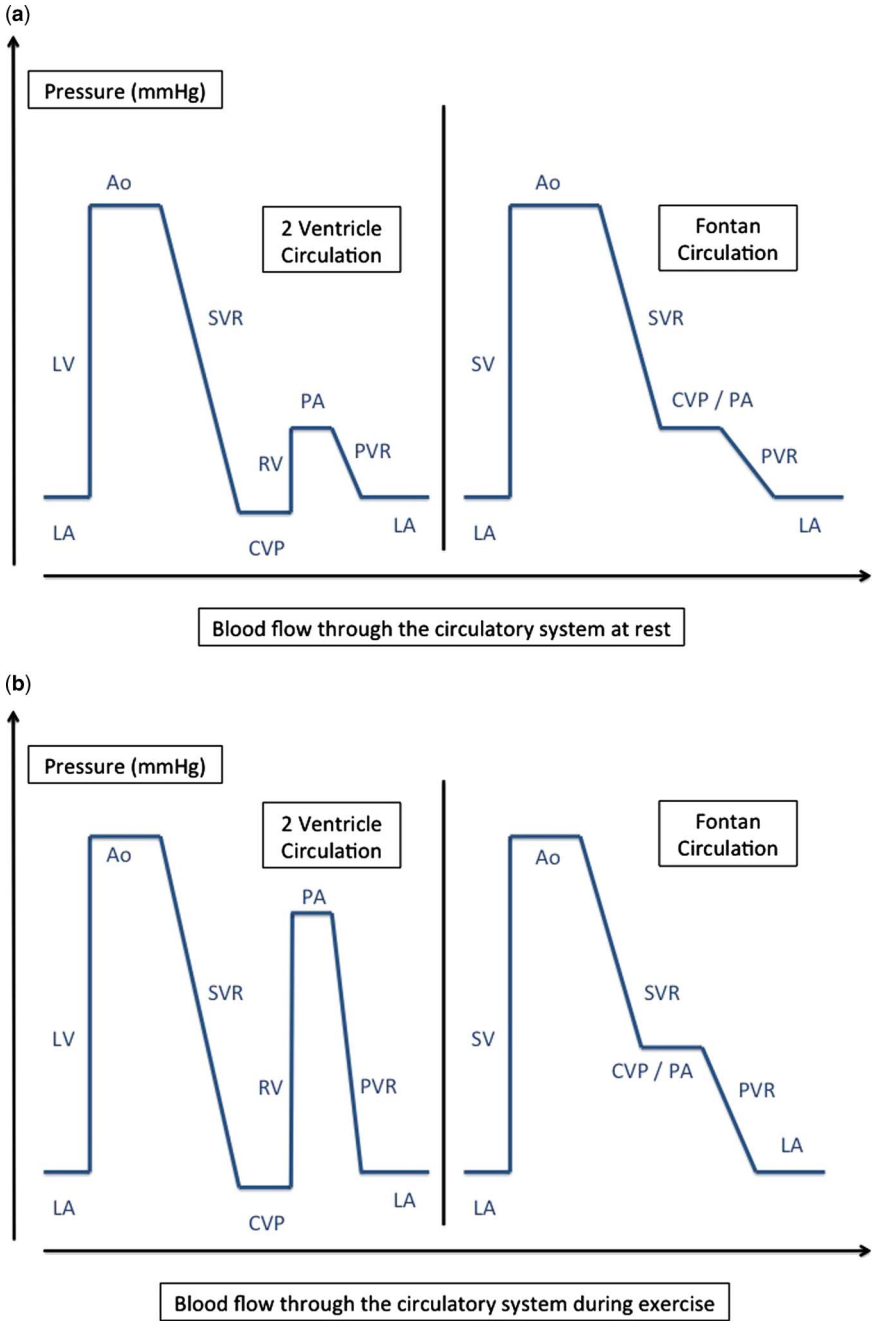


Figure 2.
*(**a**) In a two-ventricle circulation at rest, blood flow is powered through the pulmonary vascular bed by the right ventricle. In the Fontan circulation, the power is provided by central venous pressure, which may be elevated by two- to threefold above normal. At rest, the difference in cardiac output may not be perceptible. (**b**) In a two-ventricle circulation during exercise, the right ventricle powers additional cardiac output across the pulmonary vascular bed and the pulmonary arterial systolic pressure may reach nearly 50 mmHg. In the Fontan circulation, the ability to drive blood through the pulmonary vascular bed is limited by the inability to raise central venous pressure beyond a threshold value. Although cardiac output can increase by up to 500% in a normal two-ventricle circulation, the ability to increase cardiac output is severely limited in the Fontan circulation.*

in maximal VO_2 are attenuated when adjusted for lean mass in these patients. Muscle hypoperfusion, atrophy and/or abnormal metabolism may also contribute to exercise intolerance in this population.[16] Although there has been little focus on lean muscle mass in the Fontan population, a recent single-centre study evaluated this specific question. In a cross-sectional analysis, 50 Fontan participants were compared with more than 700 healthy reference participants. Similar to heart failure with

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preserved ejection fraction, Fontan participants were found to have significantly lower Z-scores for whole body lean mass and leg lean mass measured by dual-energy X-ray absorptiometry, even after adjusting for diminished growth.[17] Although additional studies are needed to assess the relative contribution of lean mass deficits to exercise capacity in Fontan patients, it seems reasonable to expect that this cohort would behave in a manner similar to those with heart failure with preserved ejection fraction.

Potential interventions: central cardiovascular

Interventions to address central haemodynamic factors may help maintain maximal cardiovascular fitness of patients with this altered physiology. These include interventions to minimise power loss and ensure normal heart rate and rhythm; pharmacological therapies to minimise pulmonary vascular resistance; and pharmacological therapies to minimise ventricular end-diastolic pressure. The study of power loss is gaining traction as some institutions have started magnetic resonance imaging-based "surgical planning" before the Fontan operation in an attempt to minimise distortions in flow related to unfavourable geometry.[18] For the reasons noted above, this type of surgical planning remains in its infancy. As our ability to accurately model the Fontan circuit improves, and we learn more about changes in the circuit with growth, the usefulness of this type surgical planning may increase dramatically. At this point, the placement of stents to relieve obstructions within the Fontan pathway, and, rarely, the alteration in the geometry of the pathway in an older patient, is the primary mechanism to address inefficiencies related to power loss.

Many cardiologists will refer for pacemaker insertion in the setting of abnormal conduction or sinus node dysfunction. Although common, the benefits of these procedures on exercise performance are often not dramatic. Except in cases of severe chronotropic impairment or loss of atrioventricular synchrony, the effects of pacing are modest. This is for the reasons stated above. The ability of the pulmonary vascular bed to maintain ventricular preload limits the advantage that a superior chronotropic response might confer to maintaining cardiac output at higher workloads. Not surprisingly, the maximal heart rates during exercise observed in studies of Fontan physiology are quite consistent at about 155 to 165 bpm. This would appear to be the heart rate range where a combination of decreased pulmonary blood flow, decreased diastolic filling time and decreased ventricular diastolic function all effectively limit the ability of the Fontan physiology to increase cardiac output and hence limit further exercise.

Even less well established is the role of medical interventions to alter the efficiency of the Fontan circuit. Given the importance of pulmonary vascular resistance and ventricular end-diastolic pressure as primary causes of exercise impairment, increased attention has recently been focused on potential therapies to address these issues. With the emergence of new therapies for pulmonary hypertension, pulmonary vascular resistance has been an alluring target for intervention in the Fontan cohort. In a pilot study evaluating the impact of sildenafil on exercise capacity, an improvement was noted in measures of respiratory efficiency, and there was a trend towards an improvement in oxygen consumption at the anaerobic threshold – a finding that reached significance in those with elevated baseline levels of serum brain-type natriuretic peptide, suggestive of more impaired haemodynamics.[19] In a European study, investigators found that a single dose of 0.7 mg/kg of sildenafil led to an improvement in maximal oxygen consumption of nearly 10% relative to baseline compared with no improvement in the group that received placebo.[20] A third study indirectly evaluated the impact of sildenafil on Fontan physiology by measuring systemic arterial oxygen saturation before and after sildenafil administration. These investigators noted an improvement in oxygen levels, suggesting either an increase in pulmonary blood flow through the lungs or an improvement in ventilation-perfusion matching.[21] On the basis of the initial work with sildenafil, additional studies have been performed using the endothelin receptor antagonist bosentan. Although neither of the two studies reported to date has demonstrated an improvement in exercise capacity following bosentan administration, planning is underway for a large, prospective clinical trial.[22–24]

Although much of the recent research has focused on the impact of pulmonary vasodilators on exercise performance, the role of ventricular compliance/end-diastolic pressure is also of potential interest. To date, there is only a single study specifically evaluating the impact of modulators of systemic vascular resistance on Fontan physiology. In that study, enalapril was administered to 18 Fontan patients in a double-blinded, placebo-controlled manner.[25] Following 10 weeks of enalapril therapy, no improvement in exercise capacity relative to baseline or to placebo was detected. However, the impact of a modulator of systemic vascular resistance on ventricular compliance may be a much longer process, and the choice of the particular medication or the particular dose used in the trial may not have been optimal to fully evaluate the hypothesis.

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Potential interventions: peripheral

Emerging information regarding the influence of peripheral factors on exercise capacity in Fontan patients suggests that there may be role for exercise training in addition to pharmacological agents. Although it is well known that habitual exercise correlates with improved exercise capacity in the general population and in some populations with congenital heart disease,[26] this progressive approach is contrary to the traditional approach of limiting activity in children with univentricular heart disease for fear of straining their physiology. Not surprisingly, patients after Fontan palliation are more sedentary compared with healthy children and adolescents[27] and may not achieve daily levels of moderate to vigorous exercise recommended to prevent obesity, diabetes and atherosclerosis.[28] However, recent studies suggest that a programme of "cardiac rehabilitation" may have a profound impact on the progressive deterioration in exercise capacity that has so far characterised the Fontan population. Cordina et al[29] demonstrated an improvement in muscle mass and maximal VO_2 after 20 weeks of high-intensity resistance training in a small group of Fontan patients compared with non-exercising controls. In addition, endurance training has been shown to improve maximal VO_2 in patients with heart failure with preserved ejection fraction, primarily through peripheral mechanisms.[16,30,31] A programme of exercise training has significant appeal, particularly when compared with the costs and patient burden of pharmacological therapy. A Fontan-specific exercise programme focused on improving lean muscle mass and overall conditioning has the potential to be quite useful for many patients in whom musculoskeletal conditioning is sub-optimal.

Summary

Exercise impairment is common after the Fontan operation and is progressive starting at adolescence. The limitation in exercise capacity is more pronounced at peak levels of exercise, but is still present during more modest levels of activity. The underlying causes of exercise impairment relate to both central cardiovascular factors (oxygen delivery) and peripheral factors (oxygen extraction). Interventions to improve cardiac preload and to improve lean muscle mass may help to improve exercise capacity and, perhaps, will alter the "natural history" of the progressive decline.

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